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Reduction of Peridural Fibrosis After Lumbar Laminotomy and Discectomy in Dogs by a Resorbable Gel (ADCON-L)

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Estimations of the rate of unsatisfactory results after lumbar disc surgery range from 5% to 40% in the literature.[7,21,22,27,30] Although the causes of the "failed back surgery syndrome" can be multifactorial, many clinical investigators consider peridural fibrosis to be one of the major etiologies for this condition.[4,6,8,10,11,20–22,24,28,35] These authors have hypothesized that the peridural scar interferes with the neuromechanics of the nerve root and dura during movement of the spine and limbs.[8] Consequently, a variety of synthetic and biologic materials have been placed into the laminectomy site of experimental animals in an attempt to either prevent or limit the amount of scar formation. Silastic, bone wax, dacron, methacrylate, bone grafts, synthetic membranes, fibrin foam, free and pedicle fat grafts, sodium hyaluronate, steroids, and anti-inflammatory agents all have been tried with inconsistent results.[2,3,12,14–18,29,31,35] The most popular of these has been the free fat graft, which many surgeons currently use. The reported results with this method are variable, however, and complications have been reported.[5,14,15,19,23,33]

ADCON-L (Gliatech Inc., Cleveland, OH) is a sterile, absorbable gel matrix comprising absorbable gelatin and a carbohydrate polymer. ADCON-L or prototypes to ADCON-L have been shown to reduce the development of fibrotic peridural scars in rat, rabbit, and dog laminectomy models in previous investigations.[25,26,34] ADCON-L functions as a device providing a barrier to fibroblasts. It is biodegradable, does not incite any inflammatory reaction, and disappears completely within 21 days. In all three of those studies, however, a simple bilateral laminectomy was the surgical procedure used; a discectomy was not done. Thus, the effects that ADCON-L might have on scar formation at a discectomy site and on anular healing could not be assessed. The latter factor is a particularly important issue because of the possibility of an increased risk of recurrent herniation from a poorly healed anular defect. Further, the rat

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Exhibit L

and rabbit models used Gelfoam (UpJohn Co., Kalamazoo, MI) as the vehicle for the active compound, a method that has been discontinued. The previous rat, rabbit, and dog studies used various prototypes of ADCON-L. The present study used ADCON-L gel and specifically added a discectomy to the model in dogs to more closely approximate the typical human surgical procedure for further evaluation of the effects of ADCON-L on peridural scar formation and anular healing. Dogs were chosen for this model because they have a spinal canal large enough to permit this procedure with a low risk of neurologic injury.

■ Materials and Methods

Surgical Procedures. Seven adult mongrel dogs were used for this study. The protocol was approved by the University of Tennessee Animal Care and Use Committee. Care of the animals and procedures done were all within the provisions of the U.S. Department of Health and Human Services Public Health Service National Institutes of Health "Guide for the Care and Use of Laboratory Animals."[32] A randomized block design with each animal serving as a block was used.

The animals were sedated with a subcutaneous injection of acepromazine (0.5 mg/kg). Atropine (0.05 mg/kg) and penicillin G (30,000 units/kg) were injected subcutaneously. The cephalic vein was cannulated, and anesthesia was induced with ketamine (35 mg/kg) and xylazine (5 mg/kg) given intravenously. A maintenance infusion of lactated crystalloid solution was started and continued throughout the procedure. The animal was intubated with a cuffed endotracheal tube, and a mixture of 0.5% nitrous oxide, 1.5% halothane, and oxygen was administered to provide analgesia with a low level of anesthesia and no apnea.

The animals then were placed prone on a heating pad on the operating table. The lumbosacral area was trimmed with an electric clipper and prepped with betadine solution. The area was draped in an aseptic fashion, and a midline incision was made from L1 to L6 and carried sharply down to the lumbosacral fascia. The fascia was divided sharply, and a subperiosteal dissection was done to expose the spinous processes and lamina on the left at each level from L1 to L6. Magnification with 2.5 × loupes was used. Hemostasis was obtained with a bipolar electrocautery.

Unilateral hemilaminotomies (mean size, 6.0 mm × 12.9 mm) were performed at three levels (either L1, L3, L5 or L2, L4, L6). The laminotomy and removal of the ligamentum flavum was done with a needle-nose rongeur and a thin footplated bone punch. The nerve root at each level was exposed and gently retracted medially using a number four Penfield (Codman, Berkshire, United Kingdom) to expose the disc space. A number 11 blade was used to make an incision into the anulus fibrosus parallel to the nerve root. A number four Penfield dissector was inserted into the anulus defect to a depth of approximately 0.5 centimeter, rotated 360°, and then removed. With this maneuver, a small amount of gelatinous nucleus pulposus extruded from the defect and was removed. Gelfoam soaked in thrombin was used for hemostasis at all sites, but was removed before closure. Sites with excessive hemorrhage were not used. A rongeur was used to partially remove the spinous process at each surgical site to clearly mark that site.

Table 1. Gross Analysis Scoring System (amount)

0 = no scar
1 = small amount of scar
2 = medium amount of scar
3 = large amount of scar

Table 2. Qualitative Assessment of Scar Scoring System (Tenacity)

0 = No adhesions between dura and annulus fibrosis or no scar present to assess
1 = slight adhesions to dura; easily detached
2 = moderate adhesions; detachable by moderate traction
3 = tenacious adhesions; detachable only by sharp dissection

After the preparation of three sites, each was designated as a sham or treated site based on a preassigned random plan. The random plain ensured that the three sites underwent surgery in a random fashion rather than consecutively. The discectomy site and nerve root of treated sites received a 1-cc application of ADCON-L, which was delivered with a 16-gauge catheter affixed to a syringe. It should be noted that because inhibition of anular healing was not seen as desirable, the ADCON-L was not injected directly into the anular defect but only around it. The wounds were closed in an anatomic fashion using interrupted 2-0 Vicryl sutures to close the fascia and subcutaneous layers and by running 3-0 Dexon sutures in the subcuticular layer. Neosporin ointment was applied to the incision, and the animal was allowed to recover from anesthesia. Intramuscular meperidine (2 mg/kg) was given in the immediate postoperative period, again the next day, then every 4–6 hours as needed. An additional dose of intramuscular penicillin (30,000 units/kg) was given 24 hours after surgery. All of the animals were maintained in similar cages with free exercise permitted.

Preparation of Specimens. The animals were killed 8 weeks after the surgery with an intravenous injection of TR-61. The entire lumbar spine was removed (from T12 to S1) en bloc. The paraspinous muscles were removed, and the specimen immediately was placed in a −70 C freezer. All specimens were evaluated later in a single session after being allowed to thaw overnight. A circular saw was used to remove the spinous processes, and a total hemilaminectomy was performed during the gross evaluation to enable complete visualization of the scar formation.

Gross Evaluation. A gross anatomic assessment of scar formation was done by an observer who was blinded to the treatment that had been used. Photographs were obtained via a camera mounted on a microscope. A rating system (Tables 1 and 2) was used to assess the amount and tenacity of the anterior scarring (discectomy site) and posterior scarring (hemilaminotomy site). Each animal served as its own control, *i.e.*, the numerical ratings assigned were based on relative comparisons between all sites that had undergone surgery within each dog specimen. Results were analyzed using a Wilcoxon signed rank test.

Histology. Operative discs from two animals were cut from the spine in the axial plane, giving a total of six discs (four

Table 3. Sham: Anterior Scar

Dog No.	Site	Tenacity	Amount	Total Scar Score
1	L2	3	3	6
2	L3	3	3	6
3	L6	3	2	5
4	L5	1	1	2
5	L3	3	3	6
6	L1	3	2	5
7	L6	3	2	5
Median				5

treated sites and two sham sites). Each disc was embedded in paraffin and decalcified in 5% formic acid for 1 week. Six- to eight-micron axial sections were prepared and stained with hematoxylin and eosin (H & E), Gomori's trichrome, cresyl fast violet, and Prussian blue. The sections then were examined in a blinded fashion for scar formation at the discectomy site.

Results

Gross Evaluation

All animals were ambulatory at the time they were killed. The skin, fascial, and muscle layers had healed well in the treated and untreated sites. The median total scar score for the sites that had undergone sham treatment was five for anterior and posterior scars (Tables 3 and 4). The median total scar score for the sites treated with ADCON-L was significantly different ($P < 0.05$), with a value of zero for anterior and posterior scars (Tables 5 and 6). Thus, compared with the condition of the sites that had undergone sham treatment, ADCON-L was found to significantly reduce peridural fibrosis at the discectomy site (anterior scar) and the hemilaminotomy site (posterior scar) 8 weeks after lumbar discectomy. Not only was less scarring present, but it was less tenacious as well. Sites that had undergone sham treatment typically had thick, tenacious scars over the posterior longitudinal ligament (Figure 1A) that adhered to the dura and nerve roots (Figures 2A and 2B). In contrast, in sites treated with ADCON-L, the posterior longitudinal ligament (Figures 1B and 1C), nerve roots, and dura (Figure 2C) had no scarring. There was no significant new bone formation noted at any of the sites that had undergone surgery. The ADCON-L appeared to be essentially resorbed at the treated sites. The incision site of the posterior longitudinal ligament made at the time of surgery had completely healed in the sites treated with ADCON-L and those that had undergone sham treatment.

Table 4. Sham: Posterior Scar

Dog No.	Site	Tenacity	Amount	Total Scar Score
1	L2	3	3	6
2	L3	3	2	5
3	L6	3	2	5
4	L5	3	2	5
5	L3	3	3	6
6	L1	3	3	6
7	L6	2	2	4
Median				5

Table 5. ADCON®-L Anterior Scar

Dog No.	Site	Tenacity	Amount	Total Scar Score
1	L4	0	0	0
1	L6	0	0	0
2	L1	0	0	0
2	L5	0	0	0
3	L2	0	0	0
3	L4	0	0	0
4	L1	3	2	5
4	L3	0	0	0
5	L1	0	0	0
5	L5	0	0	0
6	L3	0	0	0
6	L6	0	0	0
7	L2	1	1	2
7	L4	1	1	2
Median				0

Histology

Axial histologic sections of the discs taken through the area of discectomy done 8 weeks earlier showed good healing of the outer portions of the anulus fibrosus in the sites treated with ADCON-L (Figure 3) and in those that had undergone sham treatment (Figure 4). Discectomy site scars were identified by the presence of focal fibrosis (Cresyl fast violet and Gomori's trichrome stains), multiple hemosiderin-bearing macrophages (H & E; Figure 3), and focally intense hemosiderin deposition seen with Prussian blue stain for iron (not shown). An adjacent area of normal anulus taken several millimeters away from the discectomy site in one of the discs that had undergone surgery is shown for comparison (Figure 5). Although no major differences were observed between the anular scars formed at the sites treated with

Table 6. ADCON®-L Posterior Scar

Dog No.	Site	Tenacity	Amount	Total Scar Score
1	L4	0	0	0
1	L6	0	0	0
2	L1	0	0	0
2	L5	1	1	2
3	L2	1	1	2
3	L4	2	1	3
4	L1	1	1	2
4	L3	1	1	2
5	L1	0	0	0
5	L5	2	2	4
6	L3	0	0	0
6	L6	0	0	0
7	L2	0	0	0
7	L4	0	0	0
Median				0

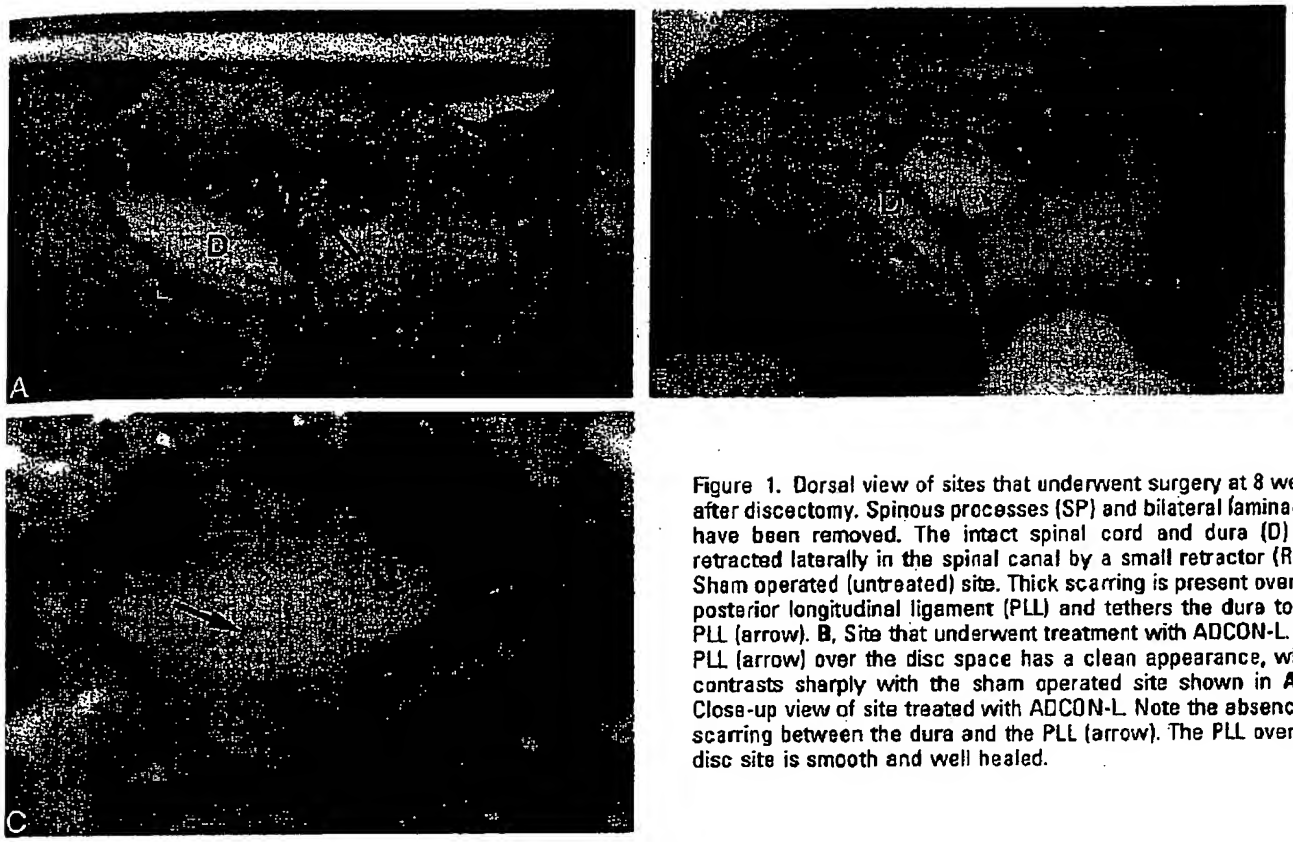

Figure 1. Dorsal view of sites that underwent surgery at 8 weeks after discectomy. Spinous processes (SP) and bilateral laminae (L) have been removed. The intact spinal cord and dura (D) are retracted laterally in the spinal canal by a small retractor (R). **A**, Sham operated (untreated) site. Thick scarring is present over the posterior longitudinal ligament (PLL) and tethers the dura to the PLL (arrow). **B**, Site that underwent treatment with ADCON-L. The PLL (arrow) over the disc space has a clean appearance, which contrasts sharply with the sham operated site shown in **A**. **C**, Close-up view of site treated with ADCON-L. Note the absence of scarring between the dura and the PLL (arrow). The PLL over the disc site is smooth and well healed.

ADCON-L and those at sites that underwent sham treatment, the latter sites did tend to show a somewhat increased density of fibroblasts and collagen in some areas (Figure 4).

■ Discussion

The findings from animal research and clinical experience suggest that multiple factors are involved in the pathogenesis of postoperative peridural scarring. Individual variability in the degree of scar formation for the identical procedure in humans and in animals account for one factor. The amount of postoperative hematoma formation has been speculated to be another important factor.[12,16,23,33] Microscopic fragments of surgical swabs and patties may have a role in the development of postoperative peridural fibrosis.[9] Peridural scar formation variability also can be attributed to laminectomy techniques, anatomic location within the vertebral column, amount of bone removed, and species and breed differences.[30] Regardless of the exact mechanisms of epidural scarring, the prevention or limitation of fibroblast migration into the wound site seems to be a key factor affecting scar formation. Previous investigations using prototypes to ADCON-L in the rat, rabbit, and dog demonstrated the efficacy of this material. However, the limited surgical procedure used in those studies (laminectomy alone) only partially replicates the surgical procedure used in humans. A discectomy was missing from those previous models, which is an important feature of the human surgical procedure.

Examination of the healing of adjacent structures in the presence of a substance that reduces scar formation is crucial to an assessment of the substance's clinical usefulness because inhibition of healing of the anulus defect theoretically could increase the risk of recurrent disc herniation. The addition of anular fenestration and discectomy to an experimental model allows for the evaluation of anular healing and of peridural scar formation at a treated site. This probably was not done in previous studies because it is very difficult to expose the nerve root and anulus fibrosus in smaller animals such as the rat or rabbit without neurologic injury. The larger spinal canal in dogs, however, allows this procedure to be accomplished safely. The dog laminectomy model has been well established in the literature, and various stages of development of the "laminectomy membrane" (epidural scar) have been well described from 1 week to 6 weeks after surgery.[1,16–18,29,31] For these reasons, a canine lumbar discectomy model was chosen for the present investigation.

In addition, a simple comparison rating system was chosen to assess scar formation because the differences observed were dramatic (as demonstrated by the gross photographs). More sophisticated surface area calcula-

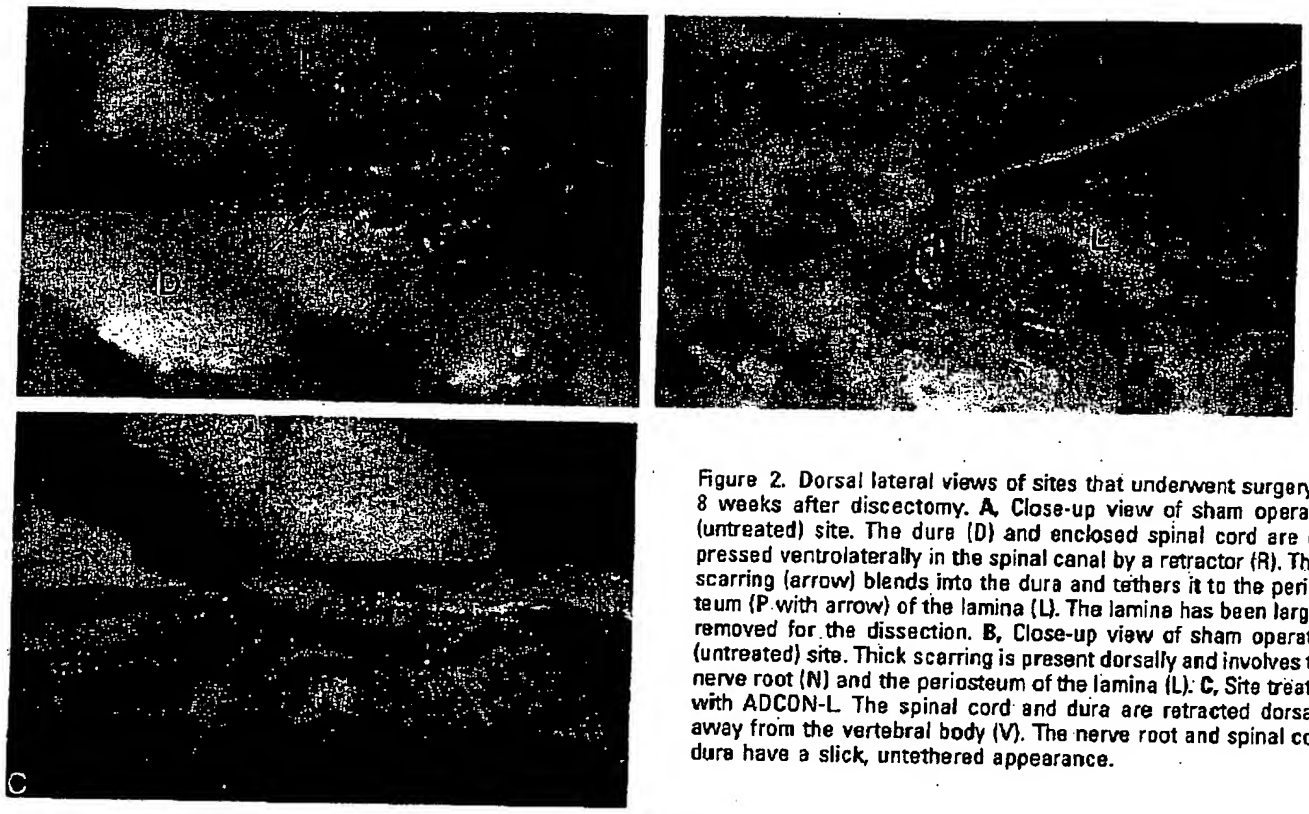

Figure 2. Dorsal lateral views of sites that underwent surgery at 8 weeks after discectomy. **A**, Close-up view of sham operated (untreated) site. The dura (D) and enclosed spinal cord are depressed ventrolaterally in the spinal canal by a retractor (R). Thick scarring (arrow) blends into the dura and tethers it to the periosteum (P with arrow) of the lamina (L). The lamina has been largely removed for the dissection. **B**, Close-up view of sham operated (untreated) site. Thick scarring is present dorsally and involves the nerve root (N) and the periosteum of the lamina (L). **C**, Site treated with ADCON-L. The spinal cord and dura are retracted dorsally away from the vertebral body (V). The nerve root and spinal cord dura have a slick, untethered appearance.

tions could have been done to make the data more objective, but the authors did not feel this was necessary. Thus, nonparametric statistics were used, and the data was summarized as median values.

The results of this study corroborate those of previous investigations using ADCON-L in the rat, rabbit, and dog with regard to reduction of posterior scar formation, and they demonstrate that anterior scarring (between the dura and the anulus) also is reduced. More importantly, the results show that anular healing after fenestration and discectomy still occurred despite decreased scar bridging between the dura and surrounding structures, which demonstrated that the antifibrotic properties are localized to the discrete area to which ADCON-L is applied. Histologic analysis of the discs that underwent sham treatment and those that underwent treatment with ADCON-L showed no significant difference in healing of the anulus. The sites treated with ADCON-L had well healed anuli, and, in fact, none of the anular fenestration scars could be identified without microscopy. The

Figure 3. Scar at the discectomy site treated with ADCON-L. Note increased cellularity from fibroblasts and hemosiderin-bearing macrophages, which are consistent with a regular healing response (arrow; hematoxylin and eosin; × 250).

Figure 4. Scar at the sham operated (untreated) site showing slightly increased numbers of fibroblasts compared with the number of fibroblasts at the site treated with ADCON-L shown in Figure 3 (H & E, × 250).

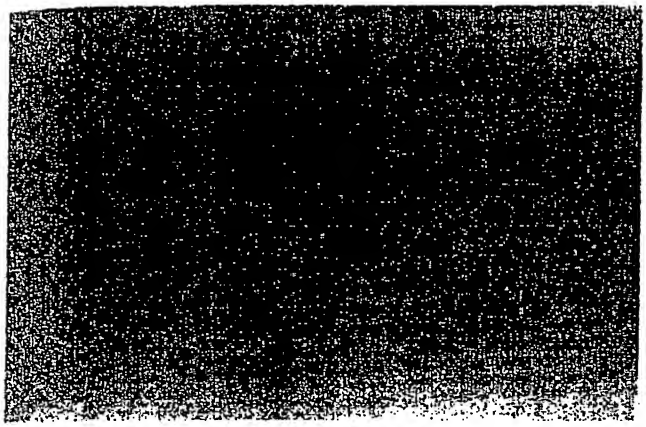

Figure 5. Normal anulus fibrosus [hematoxylin and eosin, × 250].

sites that had undergone sham treatment did appear to have a slight relative increase in the density of fibroblasts and collagen in scattered areas of the anular fenestration scar, but this subtle difference is not believed to be significant. A similar observation had been made in the rabbit study in which there appeared to be reduced collagen deposition in the peridural scar after treatment with ADCON-L.[25] The differences in the peridural scar formation noted in the rabbit study between sites that underwent treatment and those that underwent surgery with sham treatment were much more dramatic than the differences seen in anular scar formation in the present study.

The results from this investigation demonstrate that ADCON-L is effective in significantly reducing peridural scar formation anterior and posterior to the dural sac and nerve roots 8 weeks after surgery in a canine laminotomy with discectomy model. More importantly, its antifibrotic properties are localized to the discrete area to which it is applied; thus, healing of adjacent tissues is not significantly affected. This gel also might be useful in a multitude of other situations in which postoperative scar formation is a common problem, such as peripheral nerve surgery, spinal cord surgery, tendon injuries and repair, tethered cord syndrome, and myelomeningocele repair. Clinical trials using ADCON-L to determine its safety and efficacy in lumbar discectomy procedures are already in progress in Europe and in the United States.

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■ Point of View

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Since the early 1970s, fibrosis after decompression surgery of the spine, biologically a normal phenomenon, has been studied extensively, particularly its relation with recurring or persisting symptoms. The authors have restricted their study to exploring the prevention of epidural fibrosis by using an experimental animal model. The design of the study is adequate.

However, I question the validity of the rating system. They developed a qualitative rating system, but did not provide data on the reliability of this system. In particular, in the "gross analysis," a four-point scale is used without defining when the amount of scar is "small," "medium," or "large." I would like to know the interobserver and intraobserver error so that the repeatability and reproducibility can be determined.

Of greater significance is the question of whether postoperative peridural fibrosis is related to symptoms. The authors state in their introduction that fibrosis is "one of the major etiologies for this condition ('failed back surgery syndrome')." Indeed, older publications seem to indicate that that is the case.

This also has been suggested in a recent study; of 197 patients who underwent partial discectomy, 84 had extensive scarring, and 97 had less than extensive scarring, as demonstrated 6 months after surgery by gadolinium enhanced magnetic resonance imaging. Of these 181 patients, 20 demonstrated recurrent radicular pain (14 in the extensive scar group and six in the less than extensive scar group). This means that more than 10% of the patients who underwent surgery because of a lumbar radicular syndrome had a recurrent radicular syndrome within 6 months. This is very high. I wonder about the definition of radicular pain, which was not given in the article. Further, of all 84 patients with extensive scarring, only 14 demonstrated recurrent radicular pain. If extensive scarring in and of itself is thought to be the cause of the symptoms, then the number of false-positive results is far too high. This suggests a different explanation—for example, a battered nerve root. Finally, it supports the explanation given below: the scar is a coincidental finding.

Indeed, many surgeons consider fibrosis a result of healing, a normal biologic process that does not have any relation with symptoms. In patients with persisting symptoms, obviously fibrosis is observed. Theoretically two explanations are possible: the fibrosis itself may be causing symptoms, or the fibrosis is a coincidental finding and in and of itself is normal, while it is obscuring the true cause of the symptoms—for example, a sequestered disc or a battered nerve root with intraneural fibrosis. The results of more recent studies indicate the latter: Kotilainen et al[4] did not find a correlation between magnetic resonance imaging findings on the mass of epidural fibrosis and the clinical status of their patients in a pro-

spective study. Annertz et al,[1] in a prospective study, observed 16 patients after lumbar decompressive surgery by sequential magnetic resonance imaging. No correlation could be observed between the amount of fibrosis and persisting symptoms. The same authors[3] observed a poor result after repeat surgery in 35 patients of a group of 93 patients with recurring symptoms when the peridural fibrosis occurred with no other lesion. Also in a prospective study, MacKay et al[5] did not find a correlation between clinical status and socioeconomic outcome and fibrosis as observed on magnetic resonance imaging.

The National Consensus Committee of the Netherlands[2] postulated in 1995 that it has not been demonstrated that postoperative peridural scar formation is the cause of persisting or recurring back and or leg pain. Further, peridural fibrosis as such is probably not a cause of symptoms; it may indicate and obscure the real cause, however—unremoved sequestered disc, reherniated disc material, a battered nerve root, arachnoiditis, or lateral stenosis.

Postoperative fibrosis seems to be a normal biologic phenomenon correlated with healing and a result of hematoma formation. Although it is probably not the cause of persisting or recurrent symptoms in itself, it may indicate another lesion that is causing the symptoms. Technically it is easy to prevent fibrosis by meticulous atraumatic dissection as applied in microdiscectomy procedures. In those patients who bleed extensively, a method to prevent extensive scar formation seems to be indicated. In skilled surgical hands, this will rarely be needed.

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